Second, could these markers actually be reflecting glomerular inflammation? Severe interstitial inflammation is more commonly associated with class III or IV.[2] Even in this study, 11/12 (92%) class II and class V biopsies, 11/15 (73%) class III (or III + V) biopsies, and 25/37 (68%) class IV biopsies had none–mild interstitial inflammation. Thus, there is a need to adjust for severity of glomerular disease. It will be interesting to know the performance of these biomarkers looking at class III and class IV only, and with regard to the activity index (sans interstitial inflammation). Only then perhaps can we be sure that they truly represent interstitial inflammation.

Third, they have talked about biomarkers being used 'following therapy of lupus nephritis' and to 'individualize treatment decisions'. Indeed, a biomarker of interstitial inflammation (and fibrosis) may have prognostic significance.[2] However, therapy and indeed our classification systems continue to focus on glomerular inflammation.[3,4] Thus, unless interstitial inflammation itself is incorporated into classification and becomes a guide to therapy, it is unlikely that a biomarker of the same will help make treatment decisions.

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The Authors Reply: Dr Dhir[1] raises several interesting points concerning our recent publication on biomarkers of tubulointerstitial injury in lupus nephritis (LN)[2] that support the potential clinical utility of these biomarker equations. Our responses in order are as follows: (1) The biomarker equations are intended to provide a readout of kidney pathology continuously in real time to follow the response of the tubulointerstitium to therapy. (2) To examine specificity of the biomarker equations for the tubulointerstitium as opposed to the glomeruli, we tested the ability of the inflammation equation (equation 1) to determine the presence or absence of glomerular endocapillary proliferation (39% misclassified) and the presence of crescents/necrosis in >10% of glomeruli (33% misclassified). The fibrosis equation (equation 2) was used to identify biopsies having glomerulosclerosis in >25% of glomeruli (28% misclassified). These results are not unexpected, because although lesions in the glomerular and interstitial compartments are correlated, the correlations are weak to moderate.[3] However, the biomarkers used to derive equations 1 and 2 appear to also be relevant for glomerular pathologies, but will require different optimization of weighting and cutoffs. Such optimization can readily be accomplished using discriminant analysis, illustrating the applicability of this technique for biomarker development in LN and other kidney diseases. (3) Despite current glomerulocentric classifications of LN, it is well recognized that chronic tubulointerstitial damage determines long-term renal prognosis,[4] and therapeutic targeting of tubulointerstitial injury does not necessarily need to wait until classification schemes better acknowledge the importance of the tubulointerstitium.

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Laparoscopic approach for the evaluation of peritoneal injury

To the Editor: Encapsulating peritoneal sclerosis (EPS) is a severe complication of long-term peritoneal dialysis (PD) with a high mortality rate. EPS is characterized by a progressive inflammatory process resulting in the development of intra-abdominal fibrosis that envelops and constricts the viscera, thereby compromising the motility and function of the intestine, leading to partial and complete intestinal obstruction.

Takara and Ishibashi[1] reported that an endoscopy could be used for the treatment for obstructed peritoneal catheter. Here we used an endoscope for the diagnosis of EPS. We used a laparoscopic approach for the evaluation of peritoneal injury in PD patients. The peritoneum of a 64-year-old woman who